

# Cortical blindness in a patient with eclampsia: posterior reversible encephalopathy syndrome

## Introduction

Eclampsia is a well-recognized, potentially fatal complication of pregnancy that is associated with significant morbidity and causes a plethora of neurological symptoms. This article presents a case of eclampsia with complete visual loss caused by a neurological syndrome that has been well described over the last two decades.

## Discussion

Posterior reversible encephalopathy syndrome is a syndrome of radiologically evident vasogenic oedema with non-localizing neurological symptoms, such as headache, confusion, seizures and acute visual loss. It most frequently affects the posterior cerebrum, but brainstem, cerebellum and anterior cerebrum involvement have also been reported (McKinney et al, 2007).

The characteristic vasogenic oedema seen in posterior reversible encephalopathy syndrome is best visualized on T2-weighted magnetic resonance imaging sequences which show symmetrically distributed high signal intensity in the affected cerebral areas, most commonly in the parietooccipital and posterior frontal cortices and subcortical white matter (Hugonnet et al, 2012). This is distinguished from the cytotoxic oedema of infarction by isointense diffusion weighted imaging and by the reversibility of radiological changes in most cases (Covarrubias et al, 2002).

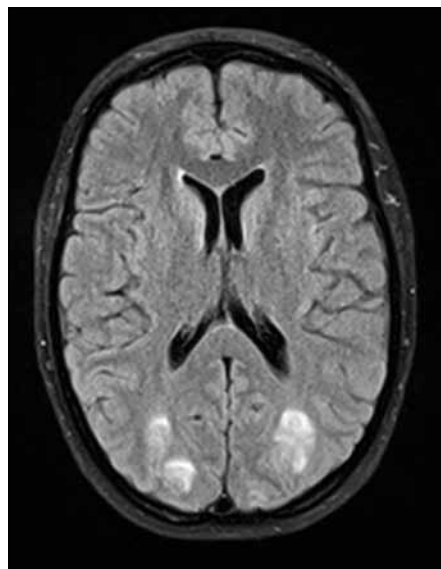
Although the pathophysiology is not fully understood, it is thought that this condition is caused by localized hyperperfusion that disrupts the blood–brain

barrier, leading to extravasation of fluid into the brain parenchyma. Hence, posterior reversible encephalopathy syndrome can be caused by a range of diseases that lead to a hypertensive crisis such as eclampsia, sepsis, acute renal failure, systemic lupus erythematosus, haemolytic uraemic syndrome and thrombotic thrombocytopenic purpura. However, it has also been reported in patients who remain normotensive and the presence of

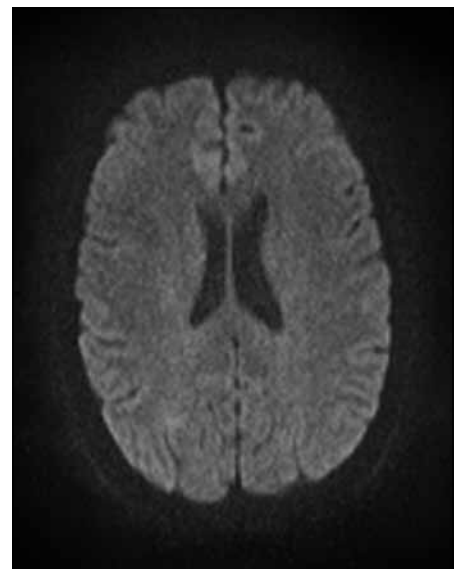
systemic toxins, such as immunosuppressant drugs or chemotherapy agents, or an allogenic bone marrow transplant, is thought to be responsible in these cases (Bartynski, 2008).

Most cases of posterior reversible encephalopathy syndrome are reversible, but cases have been described to progress to irreversible cerebral infarction and so it is important that any underlying cause is identified and promptly treated. [BJHM](#)

**Figure 1.** T2-weighted magnetic resonance imaging with FLAIR (fluid-attenuated inversion recovery) shows high signal intensity in the occipital subcortical white matter, which explains the patient's cortical blindness.



**Figure 2.** No perfusion defect is seen on diffusion weighted magnetic resonance imaging sequences, which is characteristic of the vasogenic oedema of posterior reversible encephalopathy syndrome.



## Case Report

A 42-year-old multigravida woman presented to the local hospital antenatal service at 35 weeks pregnant with acute onset bifrontal headache and altered vision. Neurological examination and fundoscopy was normal, but her blood pressure was raised at 170/108 mmHg and bedside urinalysis was strongly positive for protein and blood. The patient was diagnosed with pre-eclampsia and admitted for control of her blood pressure. Within hours the patient deteriorated, developing complete loss of vision and generalized tonic-clonic seizures. Emergency caesarean section was performed and the patient's baby was successfully delivered.

Urgent magnetic resonance imaging of the brain revealed high subcortical signal in both occipital lobes on FLAIR (fluid-attenuated inversion recovery) sequences (Figure 1), but no corresponding perfusion defect on diffusion-weighted imaging (Figure 2). This is characteristic of posterior reversible encephalopathy syndrome, which was subsequently diagnosed. The patient was started on oral labetalol for blood pressure control, and made a full recovery over 48 hours with the complete return of her vision.

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Bartynski WS (2008) Posterior reversible encephalopathy syndrome, part 2: controversies surrounding pathophysiology of vasogenic edema. *Am J Neuroradiol* **29**: 1043–9

Covarrubias DJ, Luetmer PH, Campeau NG (2002) Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion-weighted MR images. *Am J Neuroradiol* **23**: 1038–48

Hugonnet E, Da Ines D, Bobby H, Claise B, Petitcolin V, Lannareix V, Garcier JM (2012) Posterior reversible encephalopathy syndrome (PRES): Features on CT and MR Imaging. *Diagn Interv Imaging* **94**(1): 45–52

McKinney AM, Short J, Truitt CL, McKinney ZJ, Kozak OS, SantaCruz KS, Teksam M (2007) Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. *Am J Roentgenol* **189**: 904–12

## LEARNING POINTS

- Posterior reversible encephalopathy syndrome can occur as a consequence of arterial hypertension, pregnancy, immunosuppressant therapy, renal failure or systemic inflammatory disease.
- It is a serious condition which commonly presents with neurological symptoms such as visual disturbance, headache and seizures.
- If suspected early magnetic resonance imaging should be obtained to assess for any perfusion defect on diffusion weighted imaging sequences to exclude an acute stroke.
- The condition is confirmed on brain imaging, with hyperintense signal on FLAIR (fluid-attenuated inversion recovery) sequences, most typically in the posterior cerebrum.
- Prompt treatment of the underlying cause should be instated to prevent irreversible cerebral infarction, which has been reported in some cases.

## IMAGES IN MEDICINE

# Cardiac MRI of simultaneous left ventricle and left atrial appendage thrombi in a patient in sinus rhythm

A 75-year-old man was found to have triple vessel disease on coronary angiography, following an initial presentation with shortness of breath. Electrocardiogram showed sinus rhythm

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with anterior Q waves. Transthoracic echocardiogram revealed an ejection fraction of 25%. A cardiac stress magnetic resonance imaging (MRI) showed a transmural antero-septal and anterior infarct associated with thrombus in the anterior wall and left atrial appendage (*Figure 1*). There was viable myocardium in the other territories. He was commenced on anticoagulation and referred for a coronary artery bypass graft.

Left atrial appendage thrombus is an uncommon finding in the presence of sinus rhythm. It is usually associated with significant left ventricular dysfunction (as in this patient), left-sided valvular disease

or a previous history of atrial fibrillation (Agmon et al, 2002). Thrombus formation is associated with poor contraction and dilation of left atrial appendage. This demonstrates the utility of cardiac MRI in detecting unsuspected thrombi in a patient in sinus rhythm, not detected with transthoracic echocardiogram, which changed clinical management. **BJHM**

Agmon Y, Khandheria BK, Gentile F et al (2002) Clinical and echocardiographic characteristics of patients with left atrial thrombus and sinus rhythm: experience in 20 643 consecutive transesophageal echocardiographic examinations. *Circulation* **105**(1): 27–31

**Figure 1.** Cardiac magnetic resonance images showing thrombi in the left atrial appendage (white large arrow) and the anterior wall (black large arrow) of the left ventricle, and infarct in the anterior wall (small white arrows). a. Steady-state free precession cine image still post-contrast at end diastole (two-chamber view). b. Late gadolinium enhancement two-chamber view. c. Steady-state free precession cine image still post-contrast at end diastole (short-axis view). d. Late gadolinium enhancement short-axis view.

